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# U. S. Naval School of Aviation Medicine



U. S. NAVAL AIR STATION  
PENSACOLA, FLORIDA

## RESEARCH REPORT



**SPREAD OF EVOKED CORTICAL POTENTIALS  
EMORY UNIVERSITY SCHOOL OF MEDICINE  
AND**

**U. S. NAVAL SCHOOL OF AVIATION MEDICINE**

**JOINT RESEARCH REPORT**

**NO. NM 001 066.01.02**

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NAVAL AIR STATION  
PENSACOLA, FLORIDA

JOINT PROJECT REPORT

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SPREAD OF EVOKED CORTICAL POTENTIALS

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## SUMMARY AND CONCLUSIONS

Measurements of the latency and sign of the action potentials in the auditory cortex which are evoked by click stimulation have demonstrated a regularity of pattern of events. For a period of about 6 msec., no evoked activity was detected. At about this time, however, an island of positive potential appeared in the anterior suprasylvian gyrus and, at subsequent msec. intervals, wider areas showed this potential change. At about 9 msec. after stimulation, most of the ectosylvian gyri developed a negative potential. This was interrupted one or two msec. later by the change toward positivity in one or more spots near the middle of the auditory area. In subsequent msec., wider areas showed similar change, producing an expanding pattern of positive potential which died away at the borders of the auditory area. Bipolar recording demonstrated similar expanding patterns. The expanding wave traveled distances of up to 10 mm. radially from the site of origin at rates of up to 1.5 meters/second.

Extirpation of the focus or shallow cuts in the cortex tangential to the advancing wave front interrupted the positive component, but did not affect the negative potential in the middle ectosylvian gyri.

Measurement of peak latencies have demonstrated a similar cortical spread of the positive component.

The suggestion has been made that the negative component in the ectosylvian gyri has origin from diffusely projecting subcortical auditory centers and that the positive component represents transcortical propagation.

## INTRODUCTION

Studies of evoked cortical action potentials of various corticopetal systems have included statements of the location, wave shape, amplitude and, less frequently, latency of these potentials. The location has usually occupied the greatest amount of attention, while the other factors have often been included as accessory description. Latency, especially has frequently been disregarded or has been given only in terms of the maximum and minimum values.

The appearance of action potentials in different cortical locations after stimulation of a single system has been variously interpreted. It has been the basis for segmentation of the auditory cortex into primary and secondary areas, (1, 9, 10, 11) and elaborate explanations for the differences in action potentials in these two areas has been made by Bremer and Bonnet (3). In this case, as with others, the assumption has been made that there are two anatomically but functionally related areas. Similar descriptions of second or secondary cortical areas of other modalities of sense are to be found in the literature.

It is, however, implicit in much of the interpretation of the records of clinical and experimental electroencephalography that both evoked and "spontaneous" action potentials move along the surface of the cerebral cortex. Detailed studies of such activity are not abundant, but those of Brazier (2)

on sleep foci appear to corroborate the impression that wave travel does occur.

More concise descriptions of this activity have been presented by Lilly (5, 6, 7). In his work, action potentials taken from a grid of 25 points on the cortex were demonstrated on a similar grid of gaseous glow lamps, where the amplitude of the input signal influenced the brightness of the glow. This apparatus has been used to investigate the movement of activity across various fields of the cerebral cortex of animals. The nature of the indicating device has rendered interpretation difficult, since equipotential maps must be constructed from information supplied in the brightness of the lamps. There is no doubt, however, that wave forms appear to spread across the cortical surface.

These suggestions of the continuity of action potentials in the cerebral cortex cast doubt upon the validity of segmentation of functionally related areas and particularly those which are contiguous.

Because of this confusion, an attempt has been made to effect an organization of the various elements of the action potentials over a wide area of activated cortex.

In this study, the auditory cortex of the cat has been selected as the subject for examination. It was chosen because of its ready accessibility, the simplicity of delivering brief physiological stimuli and because of the controversial findings which have been reported on this system.

## EXPERIMENTAL

Eleven normal, healthy, adult cats were used. The animals were prepared by exposure of one or both lateral surfaces of the cerebral cortex under anesthesia with sodium pentobarbital.

The animals were stimulated with binaural clicks produced by activation of earphones with 100 microsecond rectangular pulses. The sound level was several times that necessary for maximum response. The stimuli were repeated at 1 second intervals.

Action potentials were collected from the pial surface with the tip of a small silver wire which acted as a monopolar electrode. A second electrode was clipped to the neck muscles or bone of the skull. A differential amplifier with a time constant of 2 seconds was used and photographic records were made from the cathode-ray oscillograph.

Since this series of experiments was designed to demonstrate the spread of cortical action potentials with single channel equipment, it was necessary to make a sequential examination of a grid of points over the entire acoustic cortex. Arrangement was made for the movement of the exploring electrode with a rack and pinion mechanism to allow considerable accuracy of placement. The points examined were 1-2 mm. apart, the exact distance being governed by interposition of blood vessels and sulci. The position of each

point was marked on an appropriate cortical map. From 3 to 7 oscillographic records were made from each point. It was found possible to complete a grid composed of 100 to 150 points in less than one hour.

Anesthetic levels were maintained as constant as possible over this period by monitoring the electrocorticogram continuously on both a cathode-ray oscillograph and an output meter. The monitored activity was taken from some point near but not within the boundaries of the auditory area. Additional pentobarbital was administered occasionally to maintain a relatively constant amount of spontaneous activity.

The animals were tested in a sound-deadened shielded room where the temperature was maintained at over 30°C, and the relative humidity about 90%. The exposed pia was moistened frequently with warm physiological saline. Grids were taken in various patterns in an attempt to control effects of changes in excitability which might occur over the period of pial exposure.

## RESULTS

Examination of action potentials from the grid of points showed that the responses were often complex, including monophasic, diphasic or triphasic waves with various temporal sequences.

Within the confines of the most active areas, the responses were predominantly positive in sign, but even here they frequently showed a negative component. Examination of the more peripheral areas showed the positive component to be of lesser amplitude and that the negative was more easily apparent.

Measurement of the earliest break from the baseline demonstrated that there were two fields of activity. One of these included most of the anterior, middle, and posterior ectosylvian gyri in which the latency was about 9 msec. and was initially negative in sign. A second area, in the anterior suprasylvian gyrus and sometimes in the anterior ectosylvian gyrus, showed an initial positive deflection with a latency of 6 or 7 msec.

The pattern of the response in the ectosylvian gyri seemed to maintain a fairly constant pattern in the deeply anesthetized animals, when a loud click was used. The sequence of events following stimulation appeared in most cases to follow the pattern of silent (latent) period, negative deflection, followed at variable periods by a positive wave, and occasionally a very late, slow, negative wave.

Further clarification of the apparently meaningless pattern of potentials was obtained by recording from a series of points taken at 1.5 mm. increments from the area of maximum positive response toward the periphery of the auditory area. Such a series is shown in Fig. 1. In this figure the stimulus artefacts are equilibrated in time. The top oscillogram was obtained from the center of greatest activity, about the superior end of the posterior ectosylvian sulcus and each successive tracing 1.5 mm. further inferior. In this figure it will be noted that each of the tracings, except for the top, shows an initial negative deflection at 9 msec. A second positive-going

change in potential is seen to occur at successively later intervals in the records from the more inferior positions. Indeed, in the case of the two most inferior points, the positive phase of activity might not be recognized unless its relationship to other stronger waves was noted. Arranged in the fashion shown in Fig. 1, however, there is an evident break which increases in latency as distance from the original center increases. When series were taken in other radii from the center of greatest activity, similar increases in latency were seen in the positive-going waves.

It may be argued that the measurement of the onset of the positive wave in Fig. 1 is actually a measure of the peak of a negative wave in some cases. The cogency of such an argument is diminished by the fact that the positive peaks also show a similar increase in latency in more peripheral records and by the statistical study of large numbers of such peak latencies (vide infra).

Except for an area in the anterior ectosylvian and suprasylvian gyri, the first consistent deflection was negative and appeared to occur in all positions in the middle and posterior ectosylvian gyri almost simultaneously. This was followed by the later development of a positive potential which interrupted the negative wave with progressively greater latencies as the periphery of the responding area was reached (Fig. 2).

Such a sequence of events was noted in the traditional auditory area only in animals under deep anesthesia and with sounds which were much louder than that necessary to produce a maximum positive potential. Under lighter anesthesia, or with less stimulus strength, the negative component was decreased or absent.

In the analysis of spread of cortical evoked potentials, measurements were made of the latencies of onset of the initial negative deflection, the first break toward positive and the peak of the positive wave. In the focus located in the anterior suprasylvian or ectosylvian gyrus, no initial negative wave appeared and the latencies of onset and peak of the positive potentials were measured.

Similar readings were made of each of several oscillograms taken at a single point. The readings were averaged to the nearest msec. and the average entered in the appropriate place on a map of the cortex of the animal. Separate maps were made for negative and positive potentials and for positive peaks. The considerable variability encountered in the peak latencies necessitated the treatment of these data in another way.

Maps made of the latencies of negative deflections have shown that there is a nearly simultaneous response over most of the ectosylvian area. This has been found to occur at 8 or 9 msec. after the stimulus. Some variation was encountered in individual animals; but, in general, the entire auditory area appeared to become negative in relation to an "indifferent" electrode at nearly the same time.

No such simultaneous activity was recorded, either in onset or peaks of the positive waves. The earliest occurrence of positive waves was in an



anterior area, situated largely on the anterior suprasylvian gyrus with some extension on the anterior ectosylvian gyrus. These responses were seen as early as 5 msec. in one animal, but were most frequently seen at 6 or 7 msec. This corresponds geographically to the area described by Mickle and Ades (8) as a polysensory area in which auditory, vestibular and somesthetic systems have been found to project.

Positive responses were not seen in the "primary" auditory area until sometime later, usually beginning about the superior end of the posterior ectosylvian sulcus at about 9 to 11 msec. after stimulation.

After entering the latencies of the positive and negative components in their appropriate places on separate cortical maps, it was found possible to construct lines which would include areas of equal latency, the lines being termed "isochrons". These are made somewhat more clear by the separation of such maps into figures showing the extent of positive and negative latencies at successive msec. intervals. These are shown in Figs. 3-18. These figures show only the boundary of the wave front at each msec. and do not indicate amplitude or duration of the wave. In these drawings, it will be seen that the earliest measurable activity occurred as a positive potential in the anterior area, which appeared at about 6 msec. During ensuing msec. the border was found to be somewhat extended. By 7 to 9 msec. a negative potential was found to appear over most of the ectosylvian gyri. This occasionally appeared as a spotty negative wave which filled in the entire area within 1 msec. About 1-2 msec. after this occurrence, one or more points were seen to change to a positive-going wave. In subsequent msec., broader areas showed such positive trends, suggesting an irregular spread from the center toward the periphery of the auditory areas. The amplitude of the positive wave was found to be greatest at the site of origin with decremental loss of amplitude as the periphery was approached. In two animals, positive waves were seen to begin later than 15 msec., and in some cases, the decrement at the periphery was to zero, with the negative never being followed by an excursion above the baseline.

The meeting of wave fronts from sources in the middle ectosylvian gyrus and the anterior suprasylvian gyrus often resulted in complex forms in the anterior ectosylvian region which were the result of combinations of waves with various phase and amplitude relationships.

The results of repeated recording from one animal are shown in Figs. 13 and 14. In this experiment, records were made from a grid of points over the responding cortex, and as soon as this grid was complete, the points were repeated, placing the electrode in an nearly the same points as possible, in the same sequence. About 45 minutes elapsed between recording from the first point of each of the two runs. The figures show that there were several differences in the pattern of potential spread. Nevertheless, the general pattern and sequence of events in the two runs were similar. The failure to repeat the exact pattern may be the result of difficulties in the method already mentioned, or of fortuitous variation due to minor and unpredictable factors.

### Results of bipolar recording

Since the problem of electrotonic spread may play a part in the recording of potentials at some distance from their source, if temporal measurements are discounted, bipolar recordings were made on two animals (A2-23 and A2-25). In each case, monopolar recordings were made immediately prior to the bipolar (Figs. 15-16 and 17-18). The bipolar electrodes used in A2-23 were a pair of silver wires, rounded at the tips, separated by 1 mm. The orientation of these tips was always in an antero-posterior direction. Animal A2-25 (Fig. 18) was examined with a concentric electrode, the outer sheath of which was a 20-gauge hypodermic needle. The electrodes in each case were moved in 1.5 or 2.0 mm. steps over the activated cortex.

Since no common reference of potential is used in bipolar recording, sign variations are not easily interpreted. In addition, potential changes which occur in phase and of the same amplitude result in null readings through the differential amplifier system. Thus, the simultaneous negative potential over the entire auditory cortex would not be reflected in the cathode-ray trace.

Records of potential spread of A2-25 (Fig. 18) demonstrate clearly the existence of two major foci of activity and their spread. The pattern of activity compares favorably with that obtained by monopolar recording (Fig. 17). The pattern of spread obtained with non-concentric electrodes in A2-23 (Fig. 16) is not readily comparable with monopolar recording from the same animal (Fig. 15). Several factors may be responsible for these differences. The anesthetic level of this animal was considerably lighter than that used in most animals and was lighter than the same animal when the monopolar records were taken. In addition, the orientation of the two electrodes with reference to the face of the oncoming cortical wave would be expected to influence the pattern obtained, i.e., the arrival of a potential wave at both electrodes simultaneously might result in complete failure to record any activity because of the differential action of the amplifiers.

In both animals, potentials recorded by the use of bipolar electrodes were not significantly smaller than those of monopolar recording, suggesting that electrotonic conduction was not responsible.

### Peak latency measurement

The apparent movement of electrical potentials previously described have all been based on the latency of the earliest change in potential sign. If, however, the activity described actually represents a difference in latency of firing of the major number of neurons and therefore of the wave peak, these also should show latency change pattern similar to that of the initiation of the positive-going wave. Such a change is demonstrated in Fig. 1.

It has been an observation of most of those who have investigated cerebral cortical action potentials that peak latencies are unreliable except when large numbers of observations have been averaged. Since the number of observations of a single spot in any one animal were insufficient in these experiments to produce a good average value of peak latency, the data have been treated in a somewhat different fashion: Measurements of the latency of peaks of positive waves have been grouped according to latency of onset of

such waves, and these peak latencies have been averaged. For example, the peak latencies for waves which had an onset at 6 msec. were grouped and averaged, regardless of the position of these points on the cortical map. The peak latencies for each of the other onset latencies were grouped and averaged in the same fashion. This treatment of the data made it possible to secure a sufficient number of peak values to make averages valid. The compilation of these data are presented in Table 1. In the table, simple averages of the peak latencies of waves with onset latencies of 6 to 17 msec. are obtained for each animal. Weighted averages of peak values were then calculated for the same onset value for all animals. These weighted averages are shown and in graphic form in Fig. 19. It will be noted that, not only is there an increase in latency of wave peaks with increasing latency in onset, but that the peaks actually appear later on a logarithmic scale. Stated in another way, the later a positive potential begins to appear on the cortical surface, the longer it takes to reach maximum amplitude. The significance of such a finding is not immediately clear. It is sufficient for the purposes of this analysis, however, to state that the onset of these waves represents a criterion which appears to be valid, as borne out by demonstration of similar peak latency changes.

#### Extirpation of epicenter of positive wave

Results of the examination of cortical spread immediately following extirpation of the focus of origin of positive activity are available on only two animals. Since determination of the position of this center depends largely upon latency measurements which must be obtained from photographic records (which were not immediately available), it was difficult to determine the extent of cortex to be removed at the time of experiment.

The results of the removal of the MEG focus are shown in A2-16 (Figs. 7-8) and A2-18 (Figs. 11-12). The spread of the positive wave is much reduced in both, while the initial negativity is still seen over the entire area. Removal of the anterior focus of positive activity is seen in A2-17 (Figs. 9-10) to have little influence upon the extent of negative reaction or spread of positive activity in the ectosylvian region.

Shallow (1 mm.) knife cuts through the cortex tangential to the expected line of advancement of the wave interrupted the progression of the positive component (Fig. 20), but did not prevent the development of the negative wave. Deeper cuts which extended to the level of the white matter did not change the character or latency of the negative wave.

#### DISCUSSION

The data presented demonstrate considerable differences in pattern of spread of evoked cortical potentials. These variations are not altogether unexpected. Individual differences in animals must be cited to account for some of these, but other factors are unquestionably active. There is no doubt that anatomical landmarks and the boundaries of functional subdivisions of the cerebral cortex have only casual correspondence. This fact, coupled with the impossibility of placing electrodes in anatomical homologues in various animals results in great discrepancies in the comparison of patterns

between animals. In addition, recording from a grid of 1-2 mm. steps probably represents only the grossest of examinations. Other factors which exert unknown degrees of influence upon the pattern are those associated with exposure of the pial surface. Trauma which is inevitable in opening the calvaria, the effects of cooling and drying, variations in anesthetic level are but a few of the insults over which only meagre control can be exerted.

Nevertheless, in spite of the obvious difficulties associated with this type of investigation, and the variation in patterns obtained, certain features occur with sufficient regularity to warrant attention: (1) There are differences in latencies of response, and these do not occur in random geographic fashion. The fact that relatively simple isochronic lines may be drawn to divide regions having latency differences of 1 msec. is sufficient evidence of this statement; (2) There is a separation of two principal types of activity, as indicated by observation of both latency and sign; (3) There appear to be at least two foci of activity which are recognized by change of potential toward positivity, one centered in the middle ectosylvian area, the other, in the anterior suprasylvian gyrus; (4) There appears to be an irregularly radial spread of this activity from each focus.

The course of cortical events following click stimulation appears to follow an organized pattern. For a period of about 6 msec. following stimulation, all parts of the cortex are free of evoked potentials. At the end of this time, a positive potential appears in a small area of the anterior suprasylvian gyrus and spreads somewhat during the next few msec. The direction of this spread is chiefly posteriorly and anteriorly. At about 9 msec., the ectosylvian area, previously silent, begins to show a negative potential over its entire surface. This is followed in one or two msec. by one or more irregular islands of positive-going potentials near the center of the ectosylvian gyri. When examined at 1 msec. intervals thereafter, positive waves are noted in increasingly larger areas about the original island or epicenter of the disturbance. The spread invades the area of negativity with decremental amplitude and increasing latency until it is lost at the periphery, occasionally leaving an uninterrupted negative wave at the extreme edges.

Monopolar recording from the cerebral cortex unquestionably causes artificial extension of borders in any mapping procedure. The use of bipolar electrodes separated by very small distances, and the use of differential amplification greatly reduces the amplitude of the record of potentials which are a result of electrotonic spread. Caution must still be exercised, however, since even with this type of recording the borders defined are, to some extent, a measure of the sensitivity of the recording instrument.

The use of concentric bipolar electrodes has shown patterns of spread of evoked potentials similar to those of monopolar recording. It is evident from these records that the spread of activity is caused by progressive cortical firing rather than the electrotonic spread of localized activity. This would indicate the presence of a neural system which allows the entire auditory cortex to be activated from a click. While the click is by no means a pure sound, neither is it composed of a spectrum of all audible frequencies. The wide distribution of action potentials following such a sound

suggests that the usual cortical handling of sounds requires or utilizes the entire auditory cortex.

The results of extirpation of various cortical areas should suggest hypotheses on the nature of this spread. It has been pointed out that it is difficult to produce appropriate lesions without having results of normal runs at hand. In addition, the spacing of grid points leaves the activity of much of the cortex in doubt. The figures (Figs. 8 and 12) showing extirpation of the middle ectosylvian focus suggest that spread is slowed or incomplete. In one case (A2-16) a new focus may have been established in the posterior ectosylvian gyrus. The results of both extirpations (A2-16 and A2-18) suggest that the continuation of activity toward the periphery of the area is a cortical rather than subcortical phenomenon, since the spread is grossly affected by the removal of central tissue. In other words, positive potentials do not appear at the edge of the extirpated area at the time they would ordinarily appear at the same position in the intact cortex.

Extirpation of the anterior focus of activity (Fig. 10) produced little effect on the spread of negative or positive potentials in the MEG area. It seems likely that if the failure of spread of positive potential in MEG were due to factors associated with trauma, removal of the anterior area would have had more effect. It was noted in this animal that extirpation of the anterior area was not complete, having omitted a small spot in the anterior MEG. This spot apparently acted as a new focus for the anterior activity, from which spread occurred.

The results of shallow knife cuts into the cortical surface were investigated (Fig. 20). In these experiments, radial series of points were examined in the intact animal. Cuts into the cortex of varying depths were made and the series repeated. It was found that cuts of 1 mm. depth resulted in an interruption of the positive wave, while the negative component was found not to be influenced by the section. Such evidence is strong indication of neuronal propagation of the transcortical positive wave, inasmuch as electrotonic conduction should have been influenced very little by such sections.

The neuron chains which are responsible for the spread of these potentials are not known. The nearly instantaneous appearance of the negative component over the entire auditory area and its failure of interruption by shallow knife cuts suggest a distribution from some subcortical source which had a diffuse cortical projection to the auditory area. The positive component appears to be a cortical phenomenon. The distances over which the wave travels from its epicenter (1 cm.) suggests that it is not the same as the potential spread demonstrated by Chiang (4) which was thought to be due to dendritic conduction in pyramidal cells. These facts could be explained more completely by chains of short neuron cells such as are found in Layer I of the cerebral cortex. The interruption of their propagation by shallow knife cuts lends substantiation to this hypothesis.

In the past there have been attempts to separate the acoustic cortex into two or more areas, most frequently called "primary" and "secondary" auditory area. These have been separated on the basis of latency (1) and frequency (10, 11) by differential effects of strychnine. A comparison of

the latencies in the middle ectosylvian gyrus and those of the posterior ectosylvian gyrus with those of Ades in his "primary" and "secondary" auditory areas shows striking similarities. A closer examination shows, however, that instead of two patterns of dissimilar activity in the two areas, there is a direct continuum of spread of the potential from the one to the other, though this is, as Ades indicated, unidirectional.

The demonstration that the entire cortical auditory area is activated in some fashion or to some degree by click stimulation suggests that further investigation should take into account the pattern of cortical activation, rather than geographical localization of frequency or amplitude, which does not satisfactorily explain the subtleties of interpretation of auditory stimuli.

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TABLE I

	A2-10	A2-11	A2-12	A2-13	A2-16	A2-17	A2-18	A2-20	A2-20	A2-23	A2-25	S.N	M. A.
			I		I	I	I	I	II	B	C	S(M)	
6	# Obs. Peak (N.M)	13 16.2 210.6	45 12.3 553.5	27 11.7 315.9	2 14.0 28.0	79 13.0 1027.0	63 10.6 667.8	26 13.3 345.8	53 10.6 561.8	39 12.3 479.7	1 12.0 12.0	353	12.0
7	# Obs. Peak (N.M)	13 17.3 224.9	13 12.6 163.8	4 12.0 48.0	18 13.0 234.0		2 14.5 29.0	36 12.0 432.0	16 13.8 220.8	37 12.3 455.1	32 12.5 400.0	171	12.9
8	# Obs. Peak (N.M)	1 16.0 16.0	1 16.0 16.0	2 15.5 31.0	6 14.3 85.8		7 13.7 95.9	10 14.2 142.0	10 13.8 142.0	1 13.1 14.0	12 13.1 157.2	39	13.8
9	# Obs. Peak (N.M)	1 16.0 16.0	1 14.0 14.0	18 15.6 280.8	6 14.0 84.0	3 21.6 64.8	1 12.0 12.0	2 15.5 31.0	6 15.6 93.6	2 14.0 28.0	24 14.7 352.8	64	15.2
10	# Obs. Peak (N.M)	9 16.2 172.8	9 16.8 172.8	39 16.8 655.2	7 15.4 107.8		18 16.2 293.4	9 15.4 138.6	2 16.5 33.0	2 16.0 32.0	28 15.6 436.0	130	16.4
11	# Obs. Peak (N.M)	10 19.3 193.0	17 20.0 340.0	35 19.6 686.0	34 16.1 547.4	5 19.0 95.0	25 17.8 445.0	49 17.5 897.5	6 16.3 97.8	20 17.2 326.0	52 16.9 878.8	310	17.5
12	# Obs. Peak (N.M)	14 20.2 272.8	28 21.2 593.6	8 26.5 212.0	16 16.1 289.8	18 21.3 383.4	47 19.1 897.7	51 19.9 1014.9	25 17.4 435.0	38 17.2 653.6	51 18.4 938.4	377	19.0
13	# Obs. Peak (N.M)	19 21.0 399.0	26 23.3 605.8	1 20.0 20.0	2 17.5 35.0	24 23.2 556.8	11 21.1 234.3	10 21.8 218.0	56 18.7 1047.2	41 18.6 762.6	5 21.8 109.0	205	20.4
14	# Obs. Peak (N.M)	8 22.2 185.6	18 25.3 455.4			9 24.4 219.6	2 27.0 54.0	22 19.8 435.6	16 19.6 313.6	4 23.0 92.0	1 21.0 21.0	80	22.2
15	# Obs. Peak (N.M)	4 27.5 110.0				8 29.1 232.8	5 22.0 110.0			2 23.5 47.0		19	26.3
16	# Obs. Peak (N.M)					6 33.8 202.8						6	33.8
17	# Obs. Peak (N.M)					5 34.0 170.0						5	34.0

LATENCY OF FIRST POSITIVE DEFLECTION IN MILLISECONDS

Table 1: Compiled data of peak latencies of the positive potentials at all positions on the cortex of all animals. # Obs. = number of observations of waves which had onset at a particular millisecond. Peak = the average peak latency of all observations for that onset latency. N.M. = number of observations times mean for that onset latency. S(N.M) = sum of N.M. M. A. = weighted average of peak latency for each millisecond of onset latency.



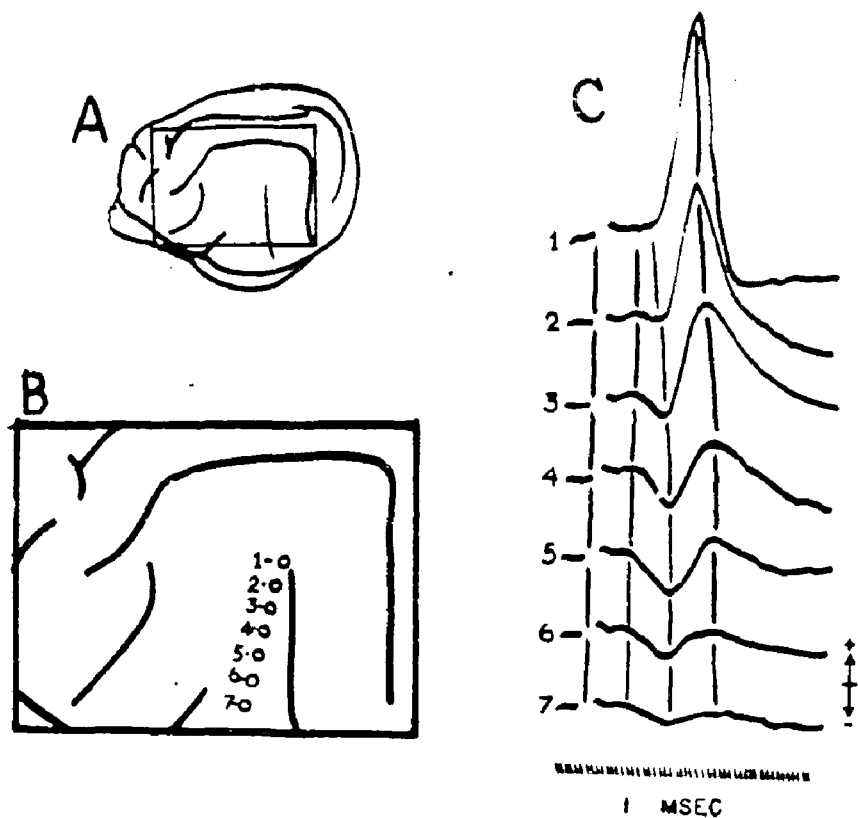


Figure 1: Diagram of auditory cortex of the cat and tracings of oscillograms taken at indicated positions. Tracings indicate simultaneous negative deflection at all points and the increasing latency of the positive component at greater distances from number 1. Increment between points approximately 1.5 mm.

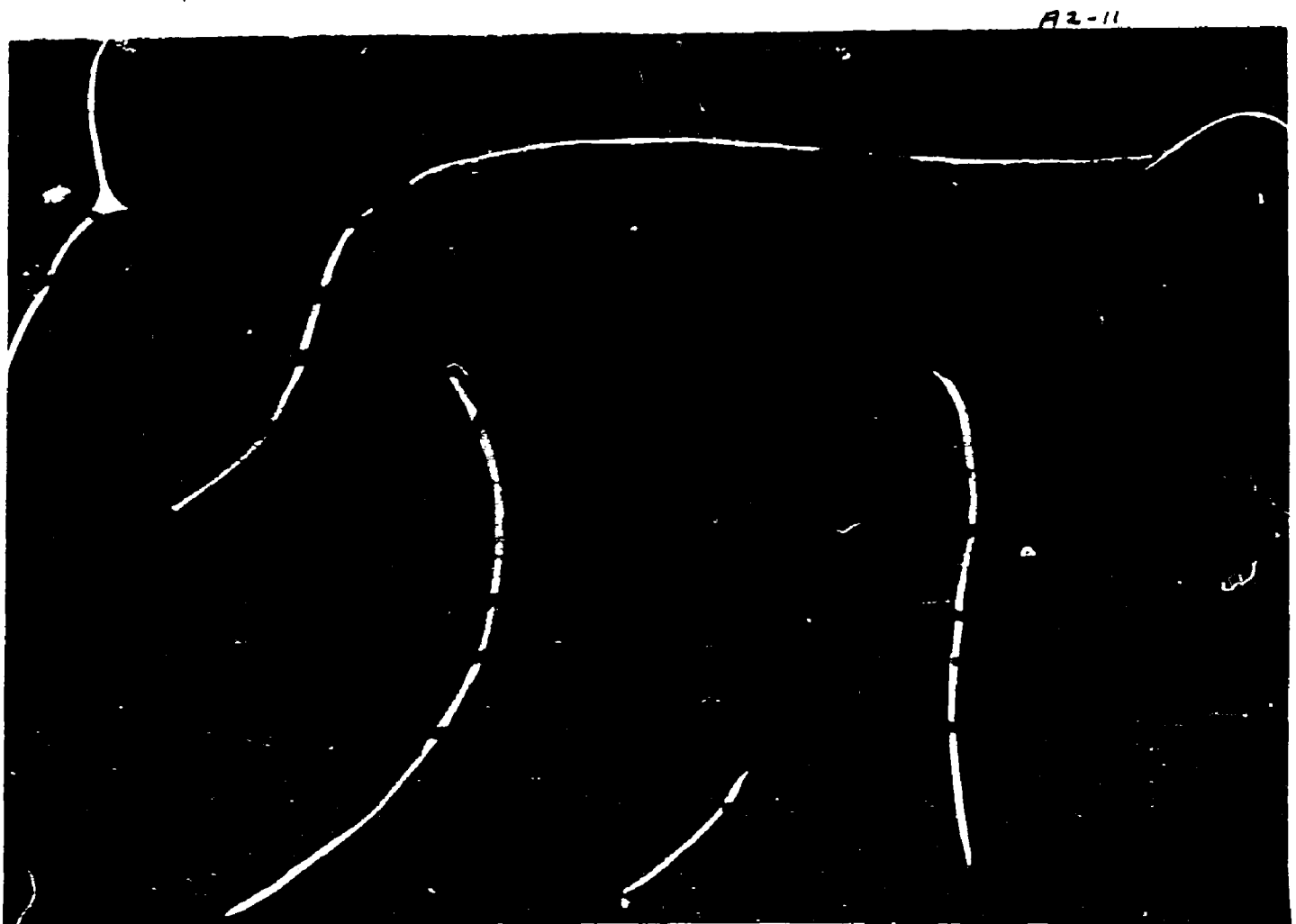


Figure 2: Sample montage of oscillograms taken at various points over the auditory cortex of the cat. Approximately 7 traces were photographed at each point, one of which is shown. Animal number A2-11.

A2 - 10

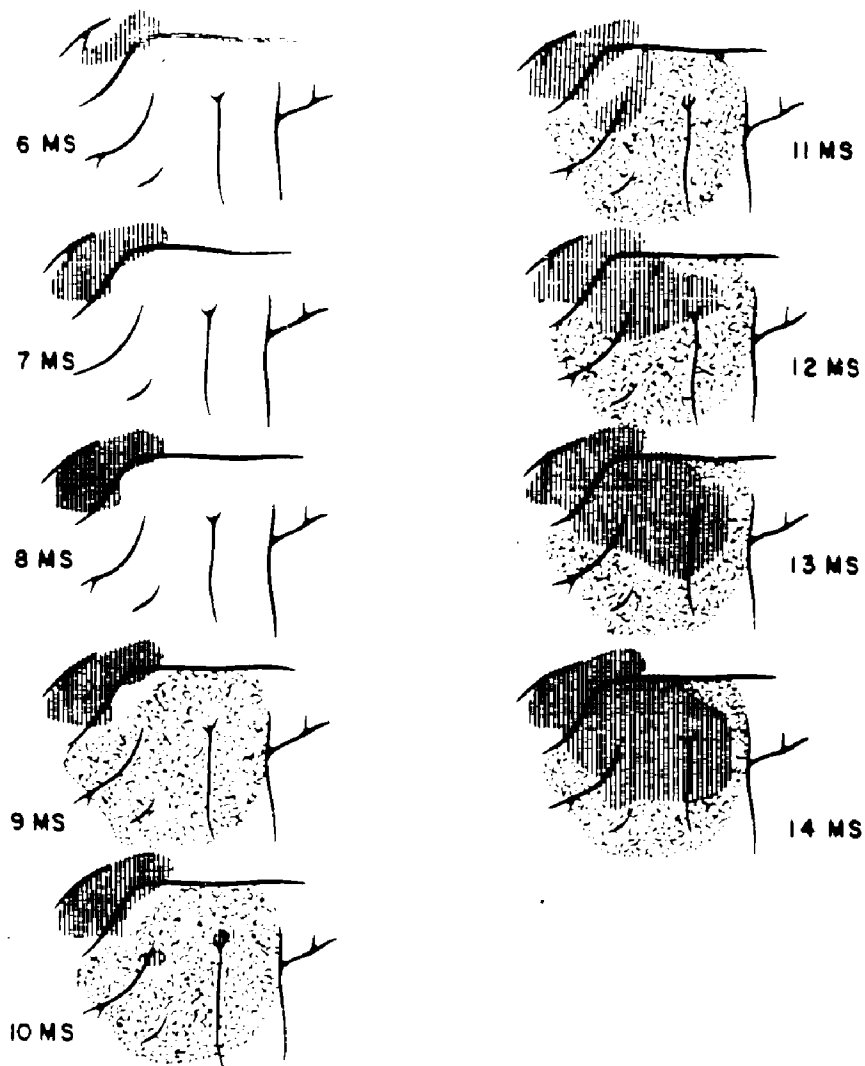


Figure 3: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-10.

A 2 - 11

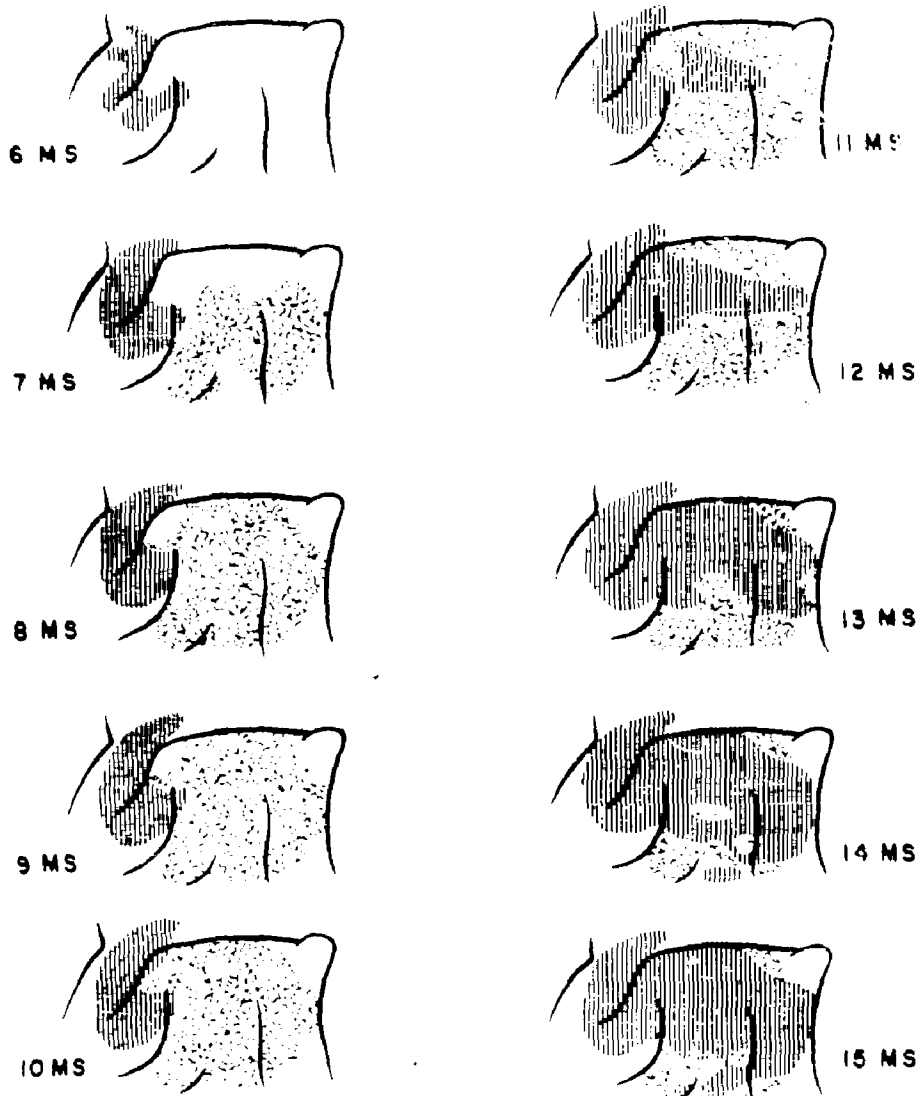


Figure 4: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-11.

A2 - 12

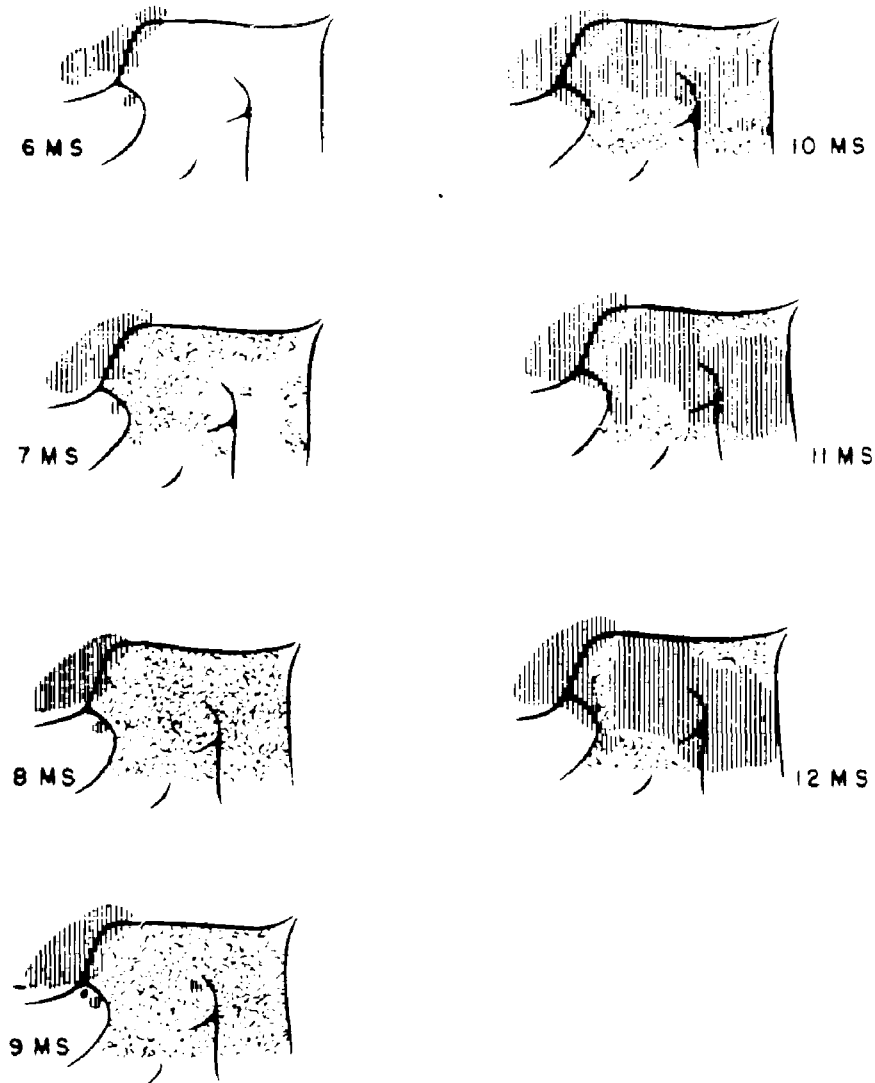


Figure 5: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-12.

A2 - 13

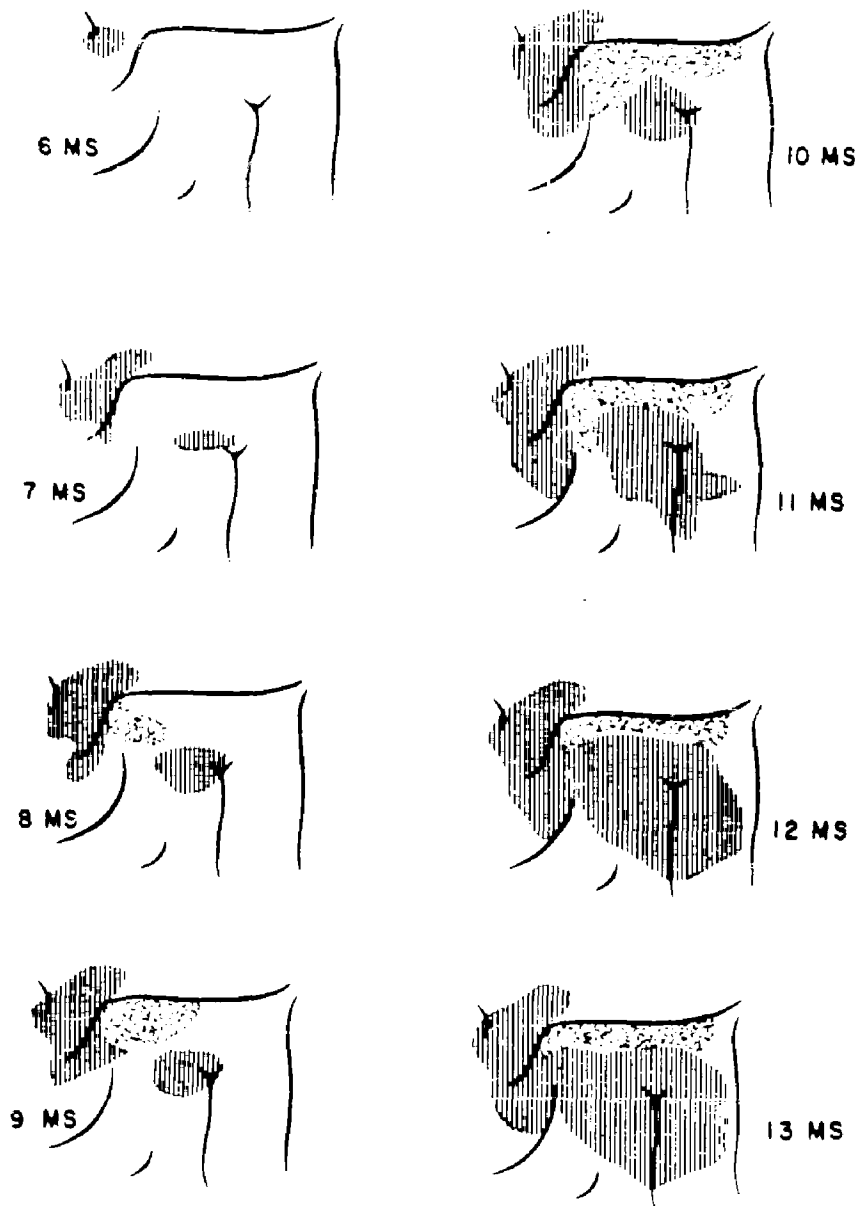


Figure 6: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-13.

A2 - 16

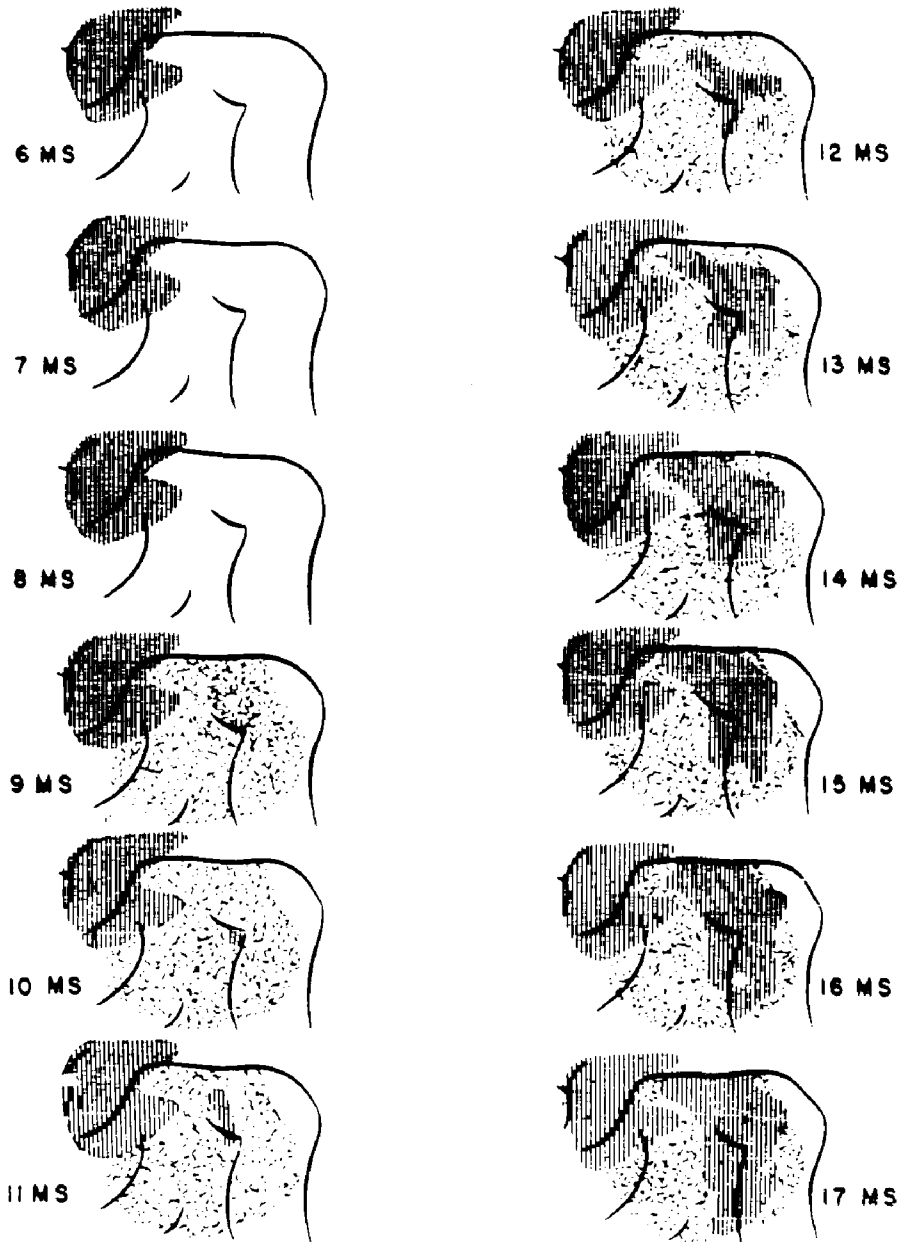


Figure 7: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-16.

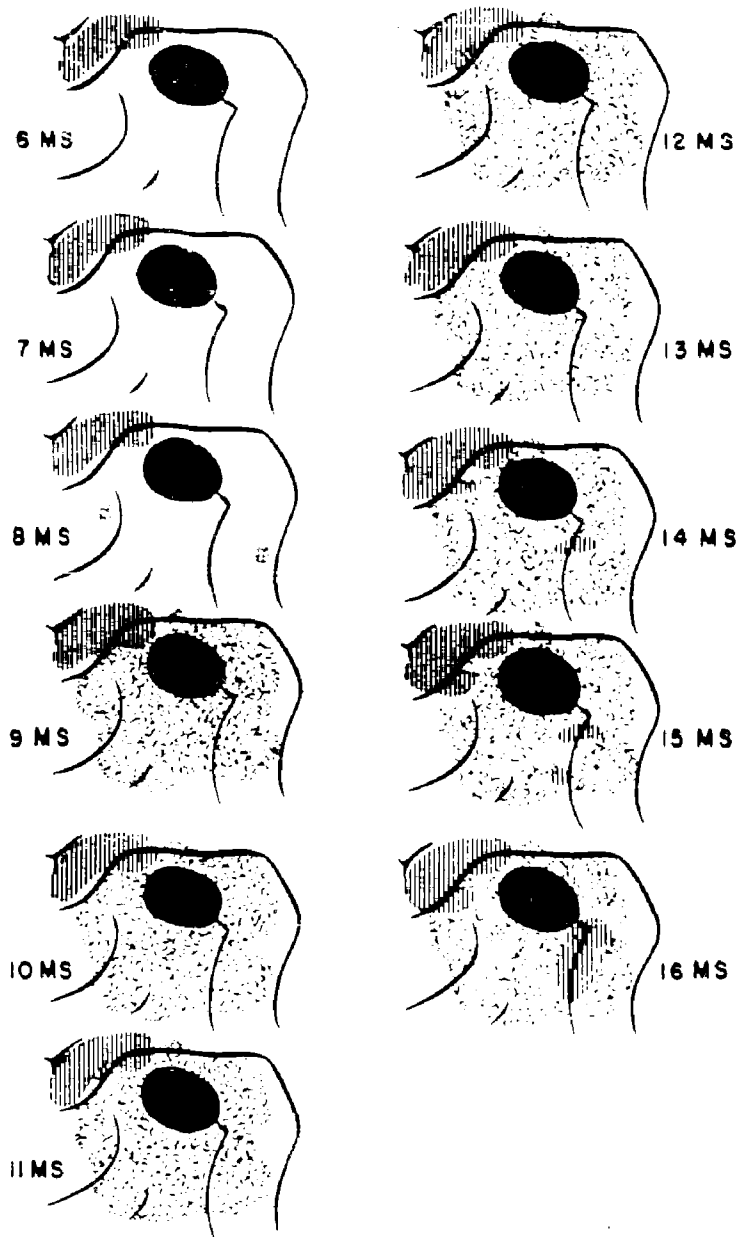


Figure 8: Map of the extent of spread of evoked potentials in the same animal shown in Figure 7, following aspiration removal of the entire cortex shown in black. Negative potentials appear over nearly the same area as before; positive spread is diminished.



A2 - 17

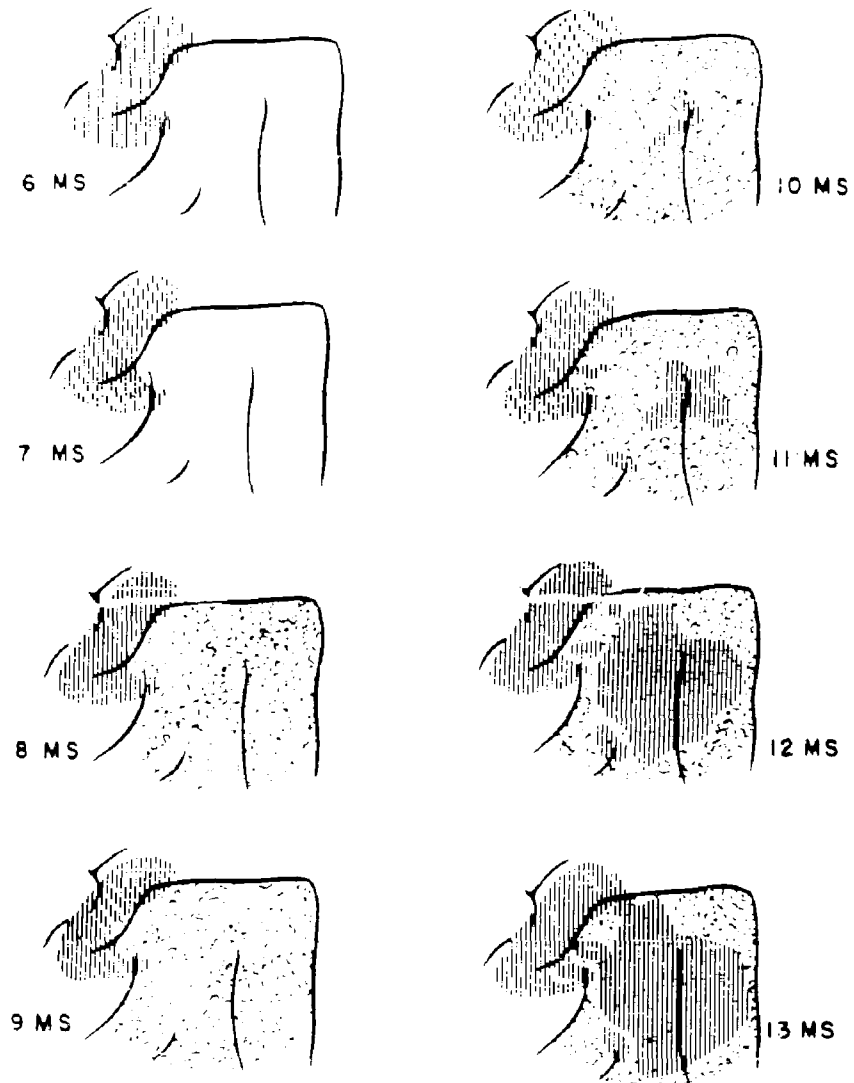


Figure 9: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-17.

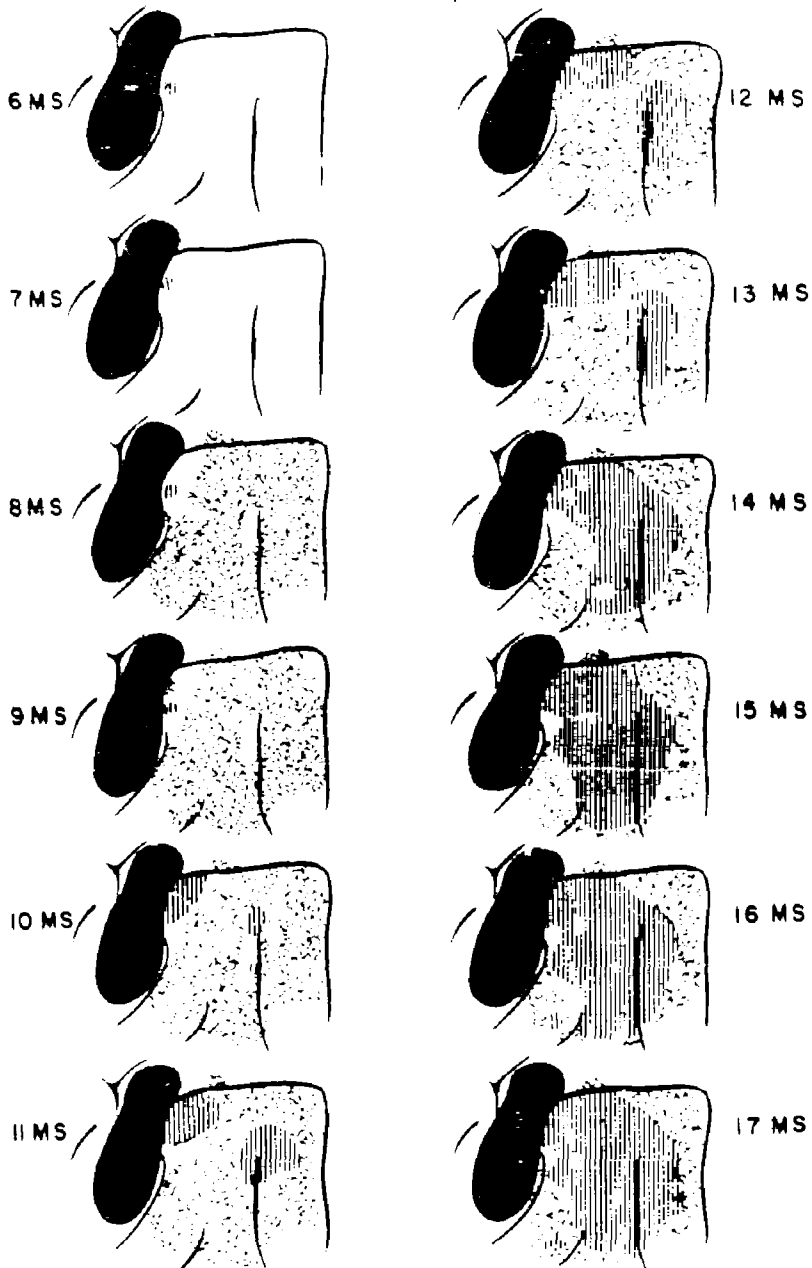


Figure 10: Map of the extent of spread of evoked potentials in the same animal shown in Figure 9, following aspiration removal of the entire cortex shown in black.

A2 - 18

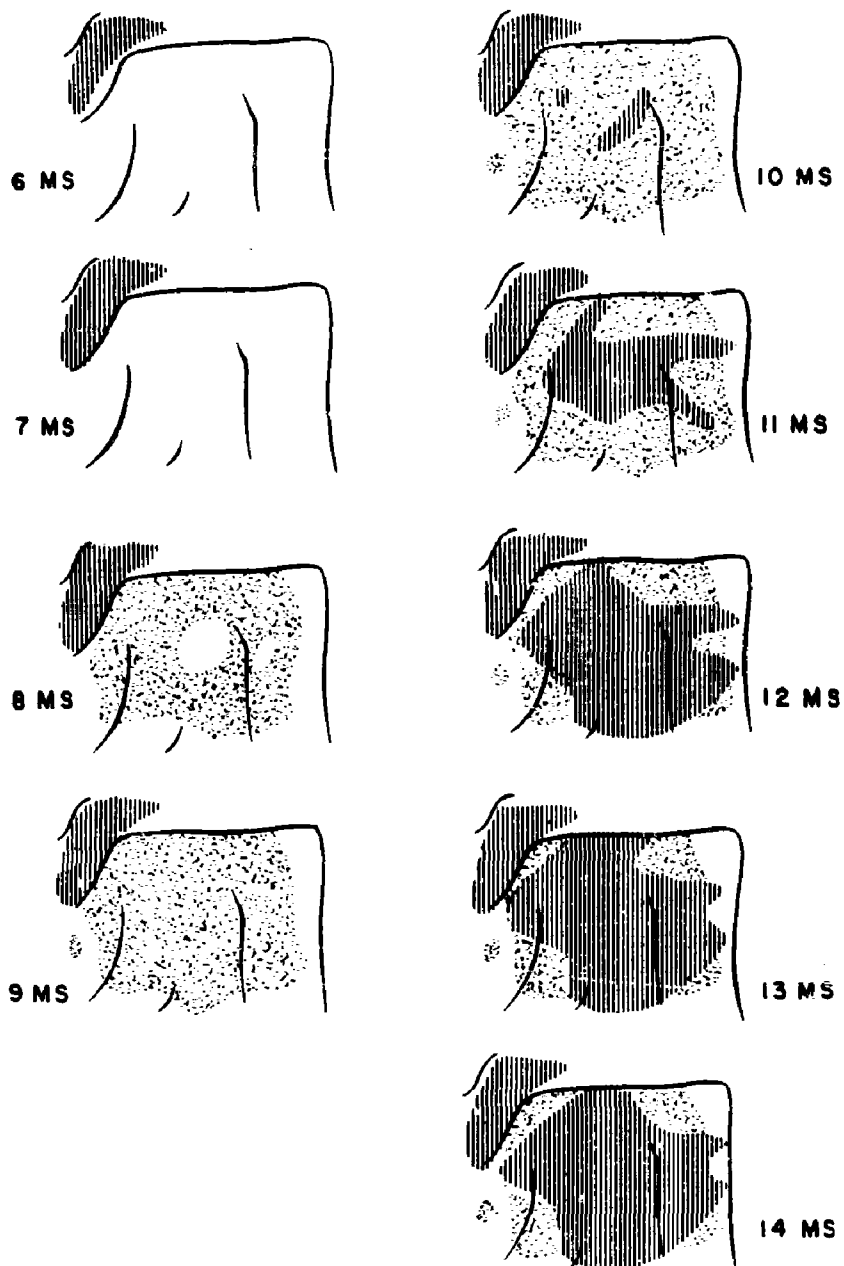


Figure 11: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-18.

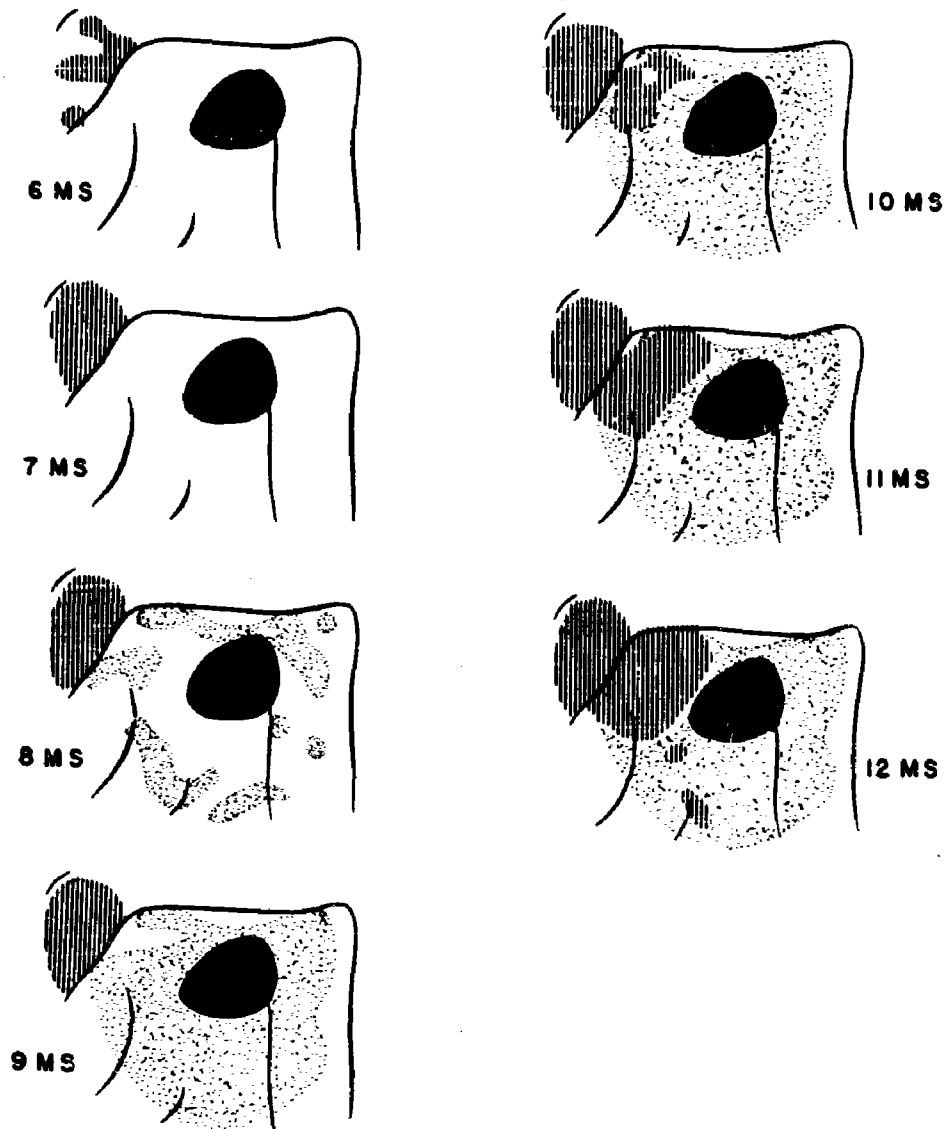


Figure 12: Map of the extent of spread of evoked potentials in the same animal shown in Figure 11, following aspiration removal of the entire cortex shown in black. Comparable experiment to that shown in Figures 7 and 8.

A2 - 201

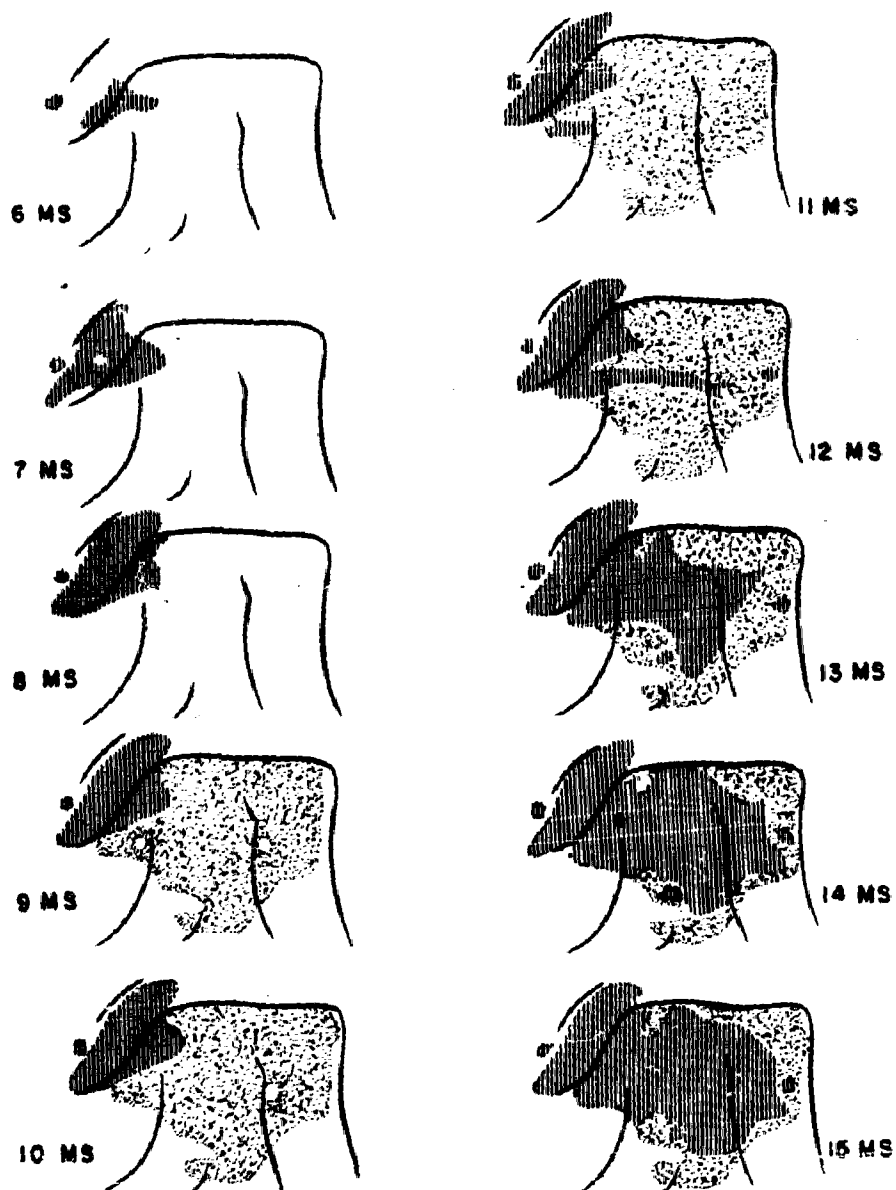


Figure 13: First of two runs on the same animal. Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-20.

A2 - 20II

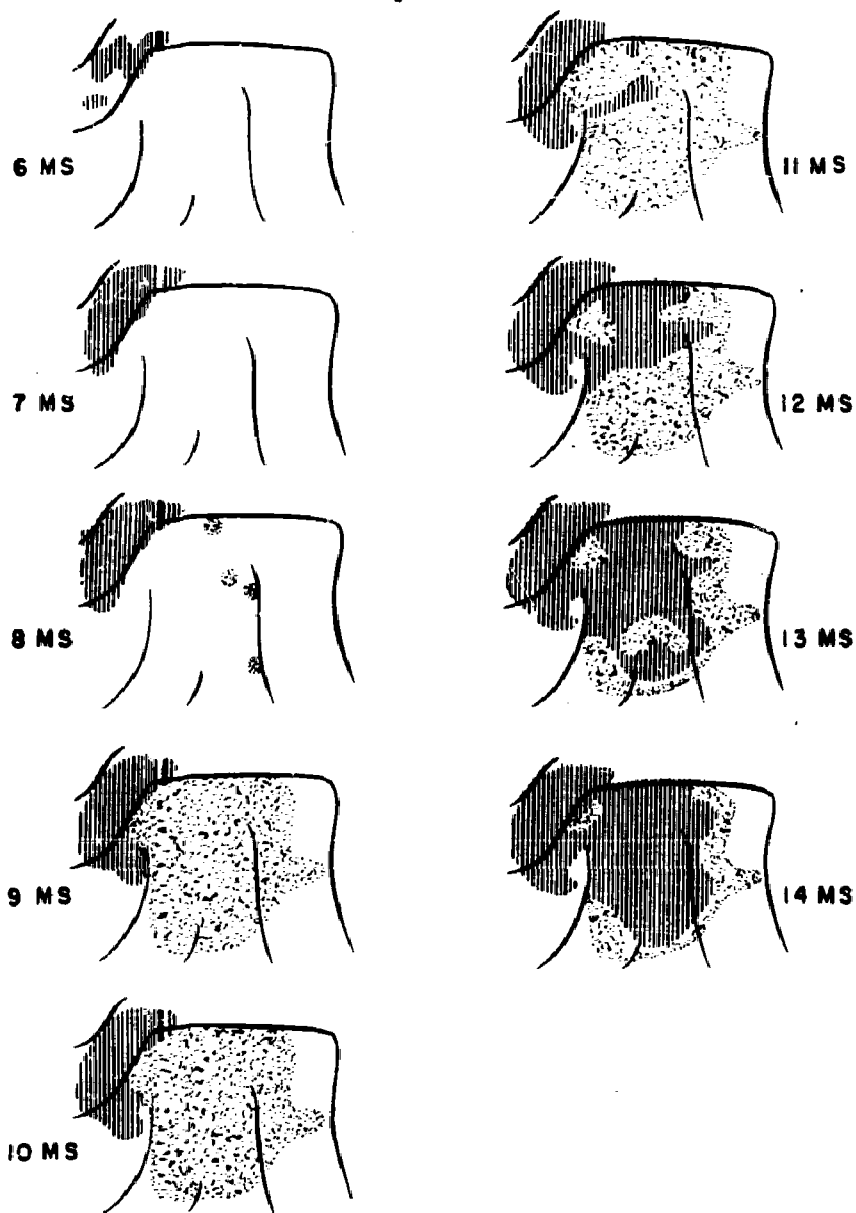


Figure 14: Second run on the same animal shown in Figure 13. Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. No operative procedures on the animal between runs; second run about 45 minutes after the first.

A2 - 23  
MONOPOLAR

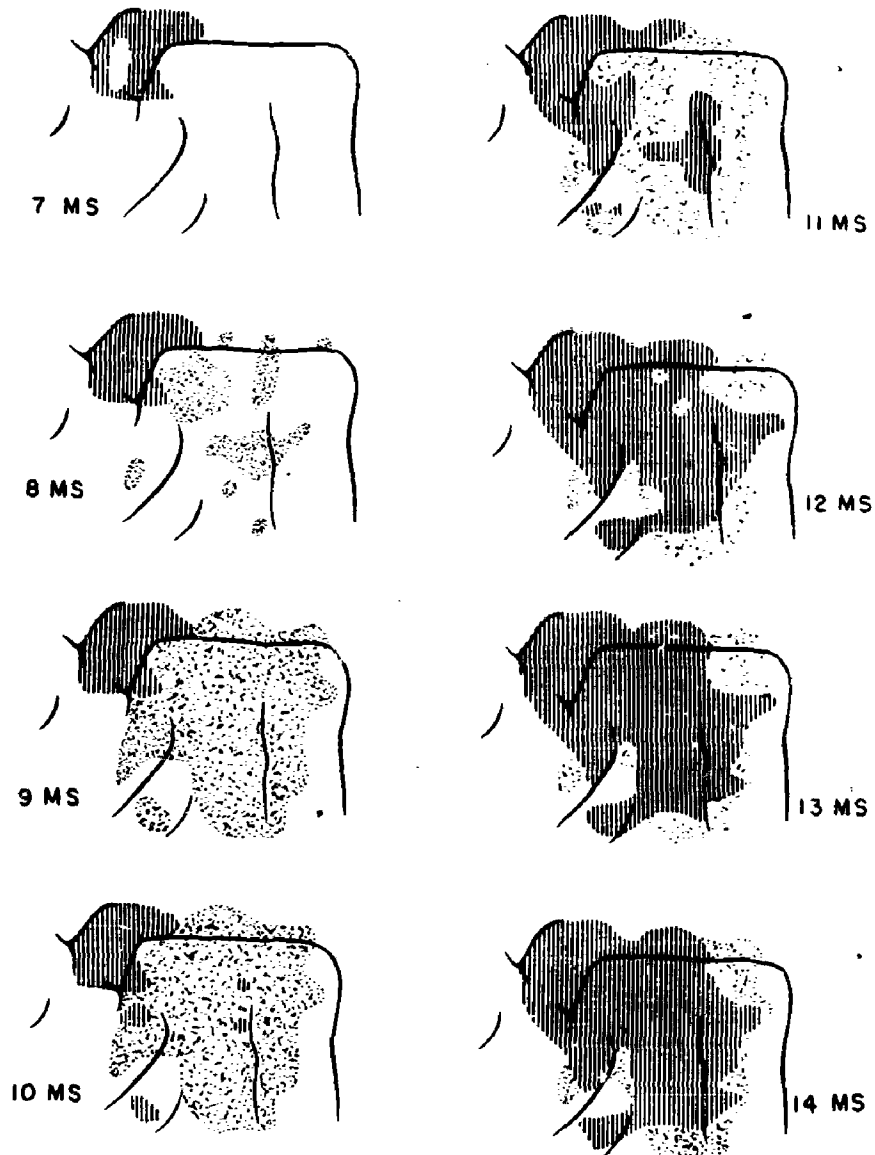


Figure 15: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-23. Monopolar exploring electrode same as all previous recordings.

A2 - 23  
BIPOLAR

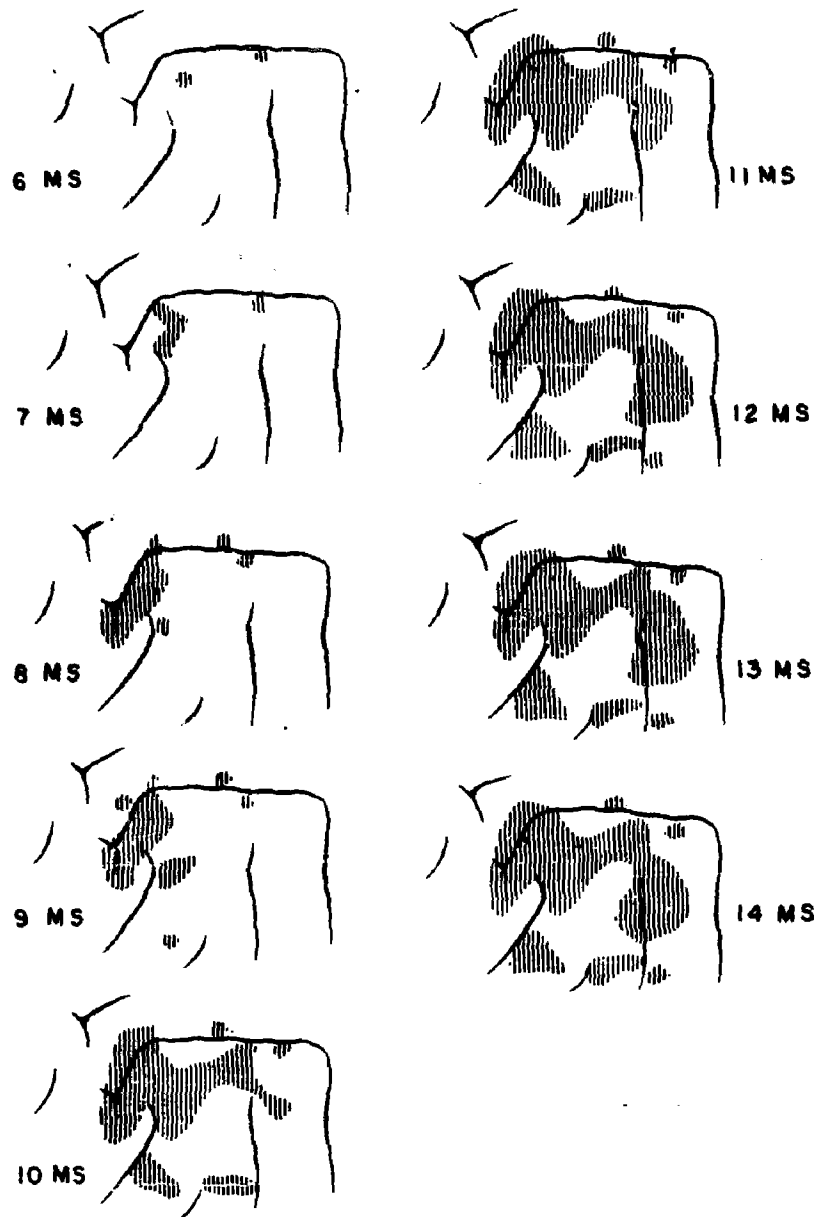


Figure 16: Same animal as shown in Figure 15. Bipolar recording from the cortex, using in-line electrodes (see text). Bar outlines indicates activity of either sign, not positive.



A2 - 25  
MONOPOLAR

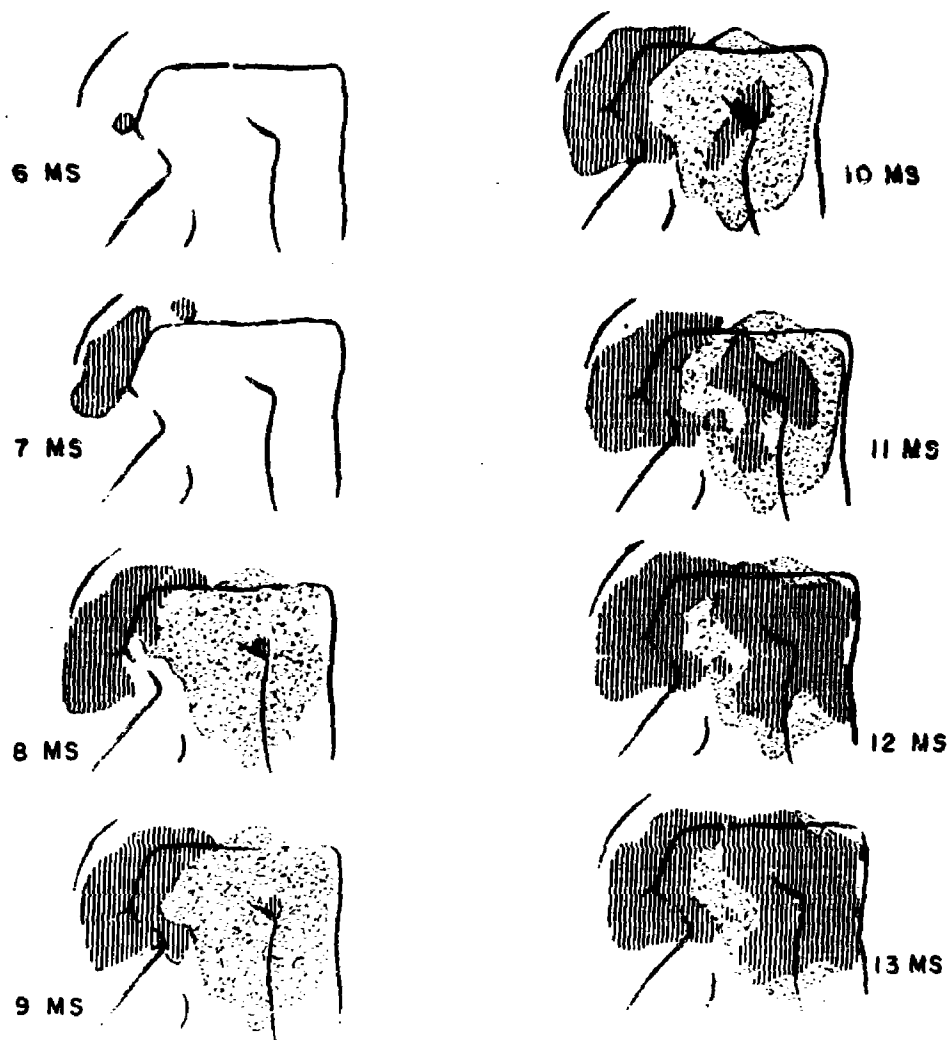


Figure 17: Maps of auditory cortex of the cat showing extent and borders of the wavefronts at millisecond intervals following stimulation. Click stimulation was used. Stipple is negative; bar outline is positive wave. Animal A2-25.

A2 - 25  
BIPOLAR

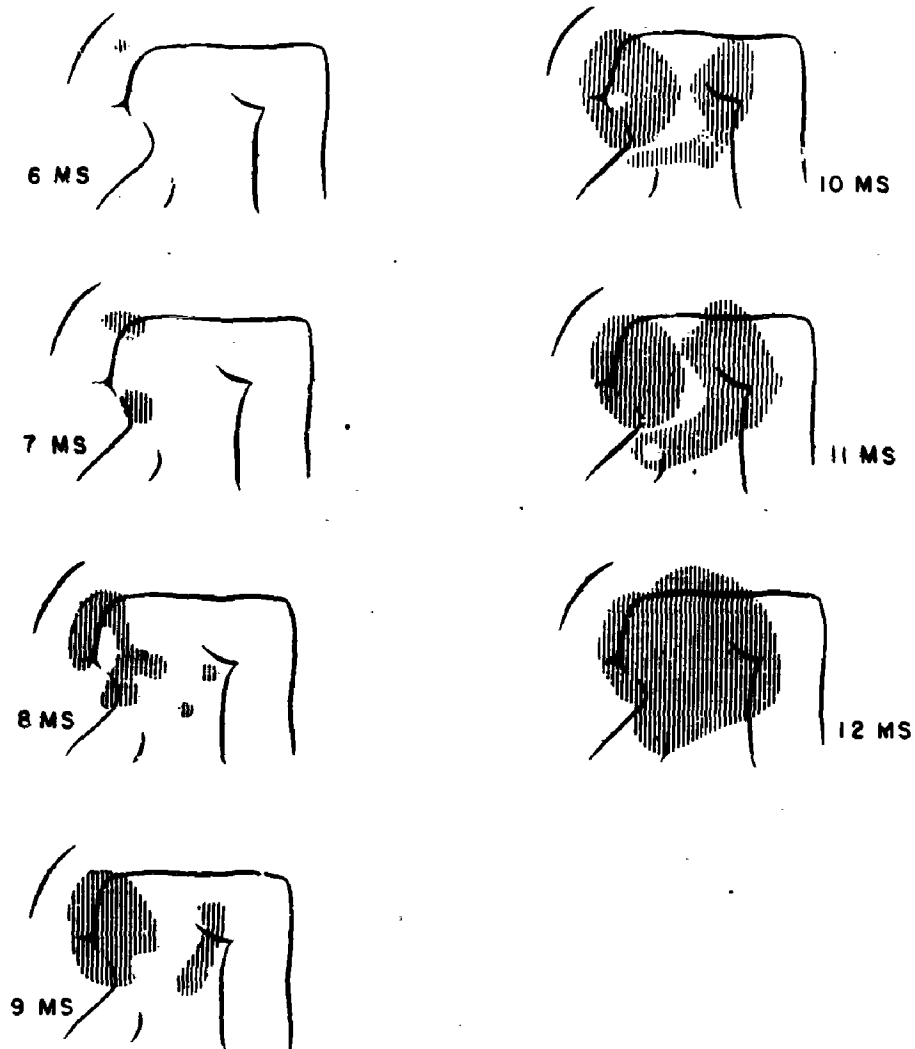


Figure 18: Same animal shown in Figure 17.  
Bipolar recording from the cortex, using con-  
centric electrode. Bar outline indicates  
activity of either sign, not positive only.

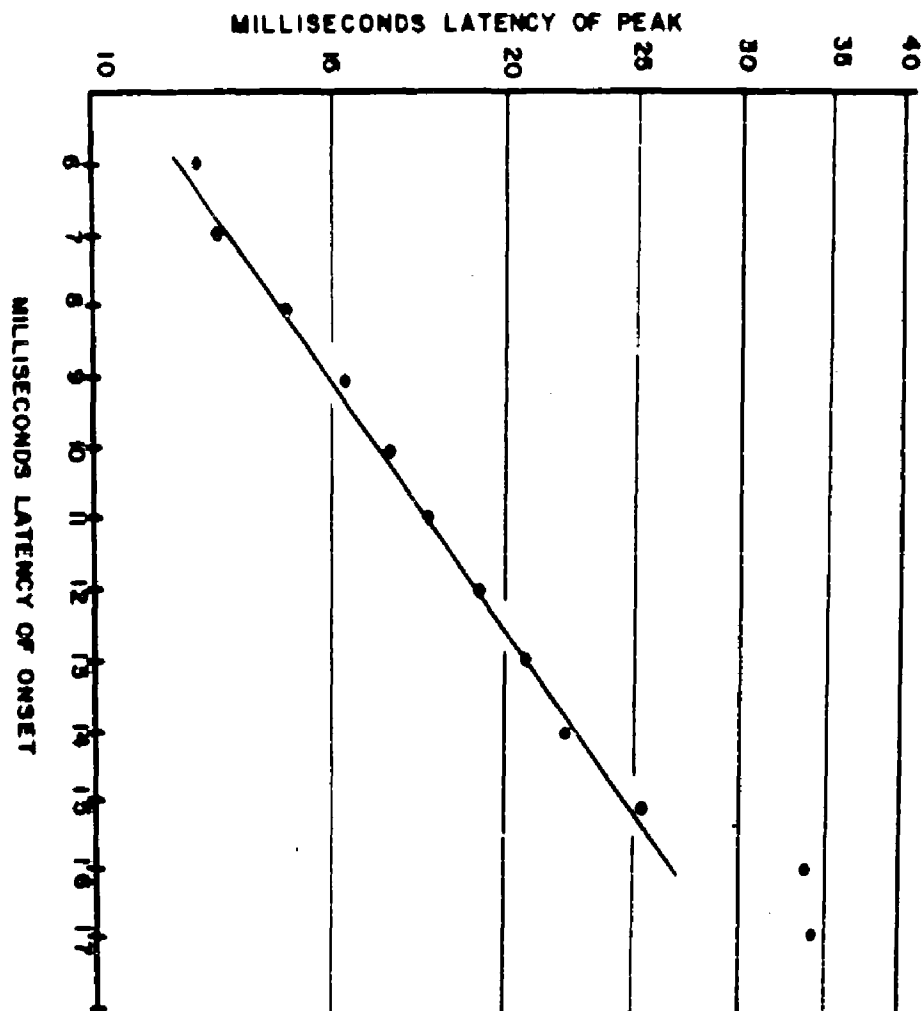
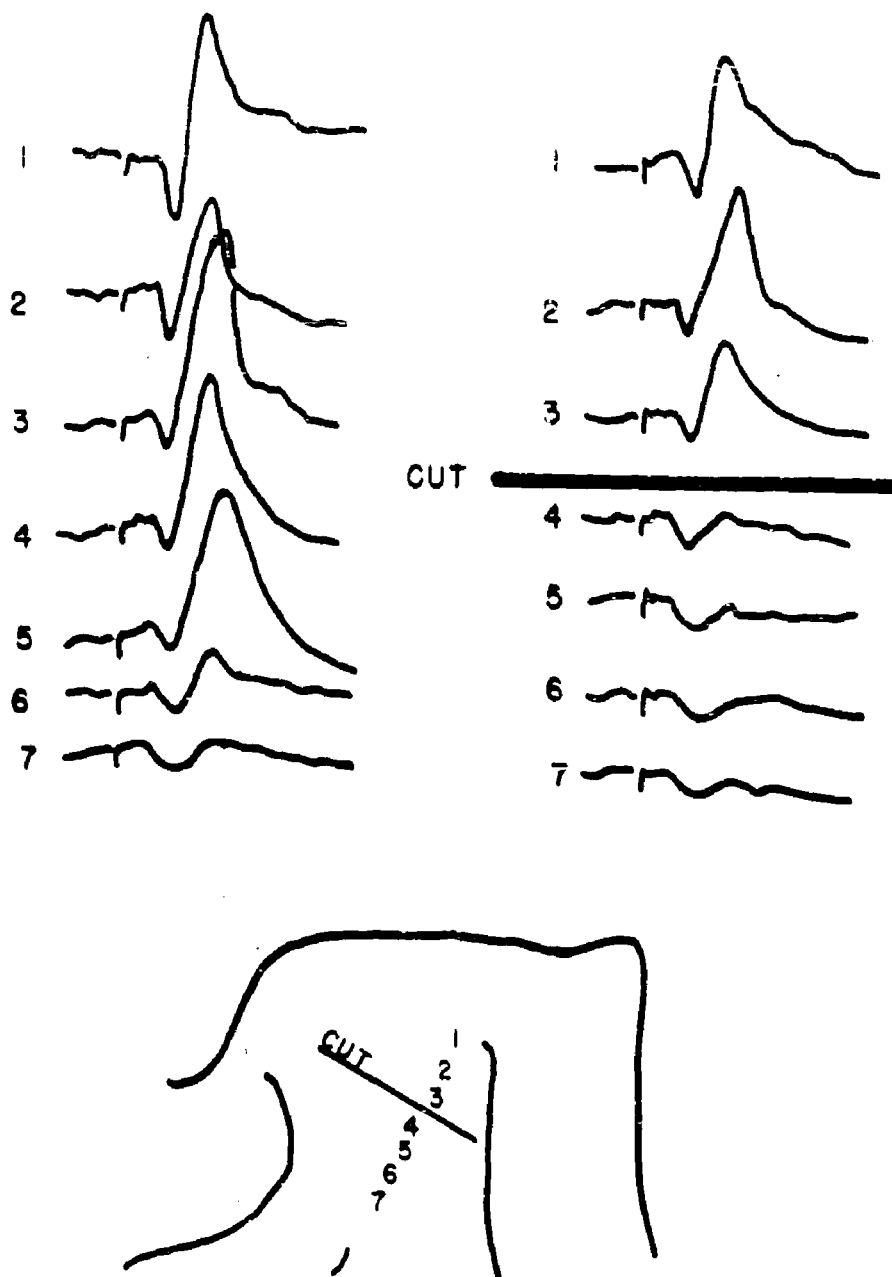


Figure 19: Graph showing the overall relationships of the onsets of positive potentials to their peak time in terms of milliseconds latency after stimulation of the end organ.



**Figure 20:** Diagram and oscillogram tracings showing the effect of a 1 mm. deep transcortical knife cut on the progress of the positive wave. Left: oscillograms taken at indicated points before knife cut. Right: oscillograms taken at the same positions after the cut between points 3 and 4.